Corporate Presentation

August 2024

SOLID BIOSCIENCES

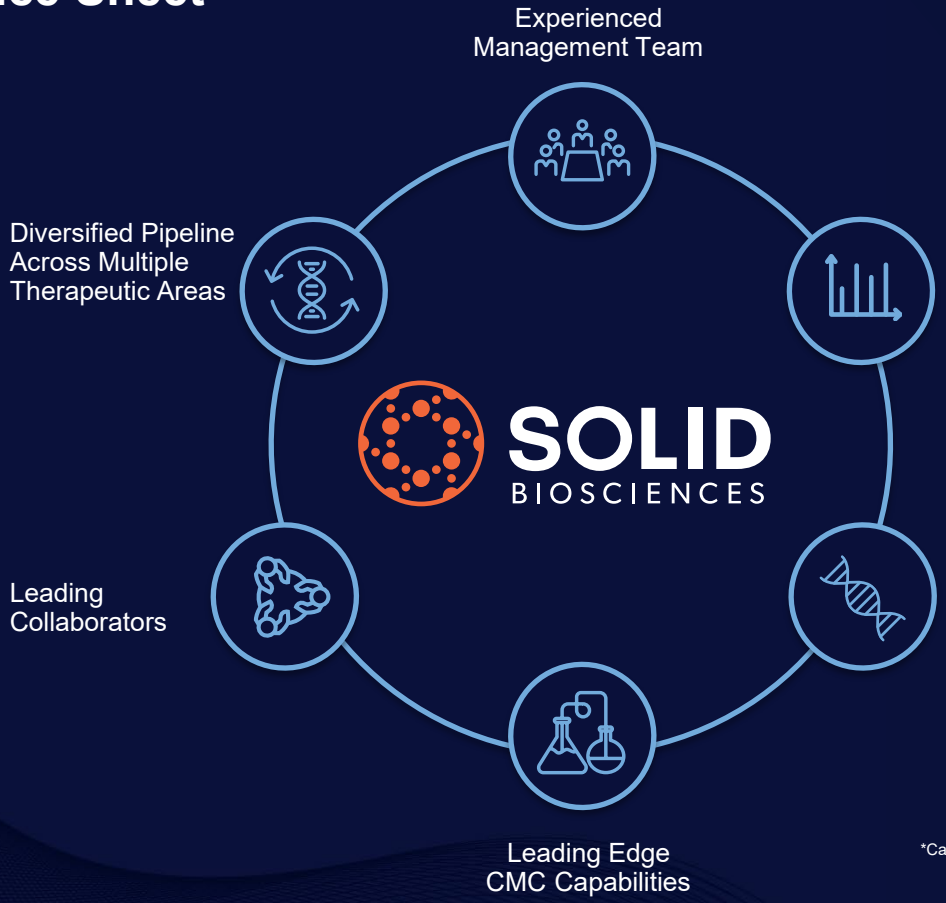
Forward Looking Statement

This presentation contains “forward-looking statements” within the meaning of the Private Securities Litigation Reform Act of 1995, including statements regarding future expectations, plans and prospects for the company; the ability to successfully achieve and execute on the company’s goals, priorities and achieve key clinical milestones; the company’s SGT-003 program, including expectations for additional CTA filings, site activations, expanded clinical development, production of additional SGT-003 GMP batches, initiation and enrollment in clinical trials, dosing, and availability of clinical trial data; the company’s expectations for submission of an IND for SGT-501 and to submit additional INDs by the end of 2026; the cash runway of the company and the sufficiency of the Company’s cash, cash equivalents, and available-for-sale securities to fund its operations; and other statements containing the words “anticipate,” “believe,” “continue,” “could,” “estimate,” “expect,” “intend,” “may,” “plan,” “potential,” “predict,” “project,” “should,” “target,” “would,” “working” and similar expressions. Any forward-looking statements are based on management’s current expectations of future events and are subject to a number of risks and uncertainties that could cause actual results to differ materially and adversely from those set forth in, or implied by, such forward-looking statements. These risks and uncertainties include, but are not limited to, risks associated with the company’s ability to advance SGT-003, SGT-501, AVB-401 and other programs and platform technologies on the timelines expected or at all; obtain and maintain necessary and desirable approvals from the FDA and other regulatory authorities; replicate in clinical trials positive results found in preclinical studies and early-stage clinical trials of the company’s product candidates; obtain, maintain or protect intellectual property rights related to its product candidates; compete successfully with other companies that are seeking to develop Duchenne and other neuromuscular and cardiac treatments and gene therapies; manage expenses; and raise the substantial additional capital needed, on the timeline necessary, to continue development of SGT-003, SGT-501, AVB-401 and other candidates, achieve its other business objectives and continue as a going concern. For a discussion of other risks and uncertainties, and other important factors, any of which could cause the company’s actual results to differ from those contained in the forward-looking statements, see the “Risk Factors” section, as well as discussions of potential risks, uncertainties and other important factors, in the company’s most recent filings with the Securities and Exchange Commission. In addition, the forward-looking statements included in this presentation represent the company’s views as of the date hereof and should not be relied upon as representing the company’s views as of any date subsequent to the date hereof. The company anticipates that subsequent events and developments will cause the company's views to change. However, while the company may elect to update these forward-looking statements at some point in the future, the company specifically disclaims any obligation to do so.

This presentation contains estimates and other statistical data made by independent parties and by us relating to market size and other data about our industry. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such data and estimates. In addition, projections, assumptions and estimates of our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertainty and risk.

SOLID BIOSCIENCES

Industry Leading Platform, Partners, Pipeline, Management, and Strong Balance Sheet



$190.3M*
as of 6/30/24

*Cash, Cash Equivalents and Available For Sale Investments

SOLID BIOSCIENCES

Clinical Stage Genetic Medicines Company Targeting Neuromuscular and Cardiac Diseases

Program	Indication	Research / Discovery	Preclinical	Phase 1/2	Milestone (anticipated)	Worldwide Rights
Neuromuscular						
SGT-003	Duchenne				FIH Data Q4 2024 [1]	✓
AVB-202-TT	FA					✓
Cardiac						
SGT-501	RYR2-Mediated CPVT				IND 1H 2025	✓
	CASQ2-Mediated CPVT					✓
AVB-401	BAG3-Mediated DCM					✓
SGT-601	TNNT2 DCM					✓
SGT-701	RBM20					✓
Platform						
Capsid Library [2]					FIH Data Q4 2024 [3]	✓

Notes: In 2020, Solid entered into a collaboration agreement with Ultragenyx for the development of UX810, a next generation Duchenne construct comprised of Solid's proprietary nNOS microdystrophin and Ultragenyx's Pinnacle™ PCL manufacturing platform for use with AAV8 and Clade E variants thereof. Solid has the option to co-fund collaboration programs in return for a profit share or increased royalty payments at proof-of-concept. 1. Initial safety and 90-day expression and functional data; 2. Capsid Library currently in NHPs, Mice and Pigs; 3. AAV-SLB101

SOLID BIOSCIENCES

Q2 2024 Corporate Update:
First-in-Human Duchenne Clinical Trial Initiated & Multiple INDs Expected Through 2026

PATIENT DOSING COMMENCED IN SGT-003 PHASE 1/2 INSPIRE DUCHENNE TRIAL	First-in-human evaluation of SGT-003 for treatment of Duchenne Muscular Dystrophy; initial data expected Q4 2024
SGT-003 WELL TOLERATED IN INITIAL PATIENTS DOSED *	Based on safety seen to date, Solid plans to expand INSPIRE DUCHENNE with additional sites in the U.S., Canada, and Europe – additional GMP batch production planned to support expansion
TARGETING SUBMISSION OF 3-4 INDs BY END OF 2026	Strategically selecting neuromuscular and cardiac diseases; CPVT IND submission expected 1H 2025
STRONG PROGRESS IN CAPSID LIBRARY OUT-LICENSING	AAV-SLB101, the proprietary capsid used in SGT-003, is now being used by 10 academic labs and 1 corporation, with additional negotiations underway

* Data reported on August 13, 2024

SOLID BIOSCIENCES

Neuromuscular Lead Program

Duchenne Muscular Dystrophy (Duchenne)

SOLID BIOSCIENCES

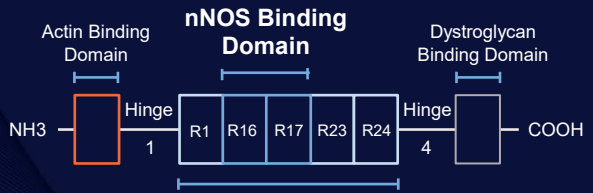
DMD

SGT-003 Utilizes an Optimized Transgene, Next Generation Capsid and Improved Manufacturing Process

Next-Generation Construct Has Shown Promising Results in Preclinical Testing

Transgene

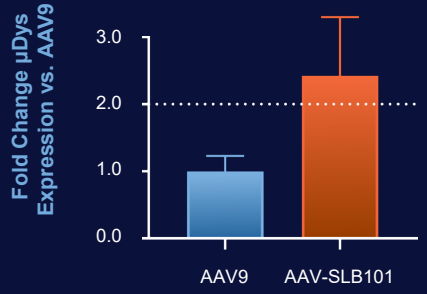
Solid's microdystrophin uniquely includes the nNOS binding domain, potentially important for prevention of activity-induced ischemia and associated muscle injury



Capsid

Rationally designed capsid with the goal of improving skeletal muscle tropism

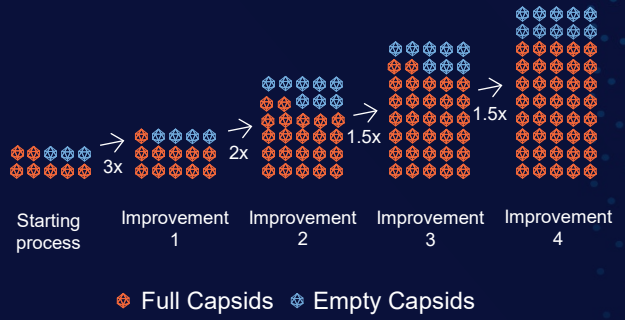
Robust µDys Expression in mdx Mouse



Manufacturing Process

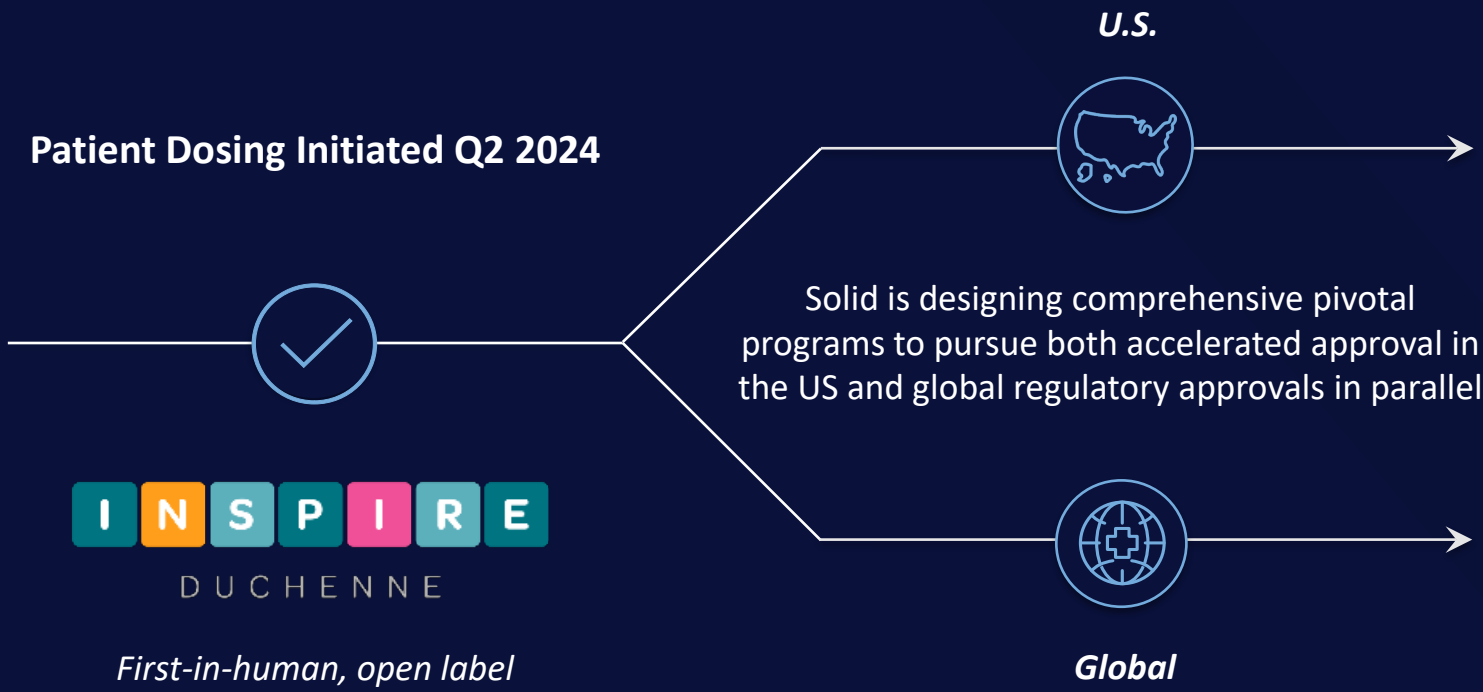
Current yields and full to empty ratios have potential to significantly reduce COGS for Duchenne and other gene therapies

Full/Empty and Yield Improvements

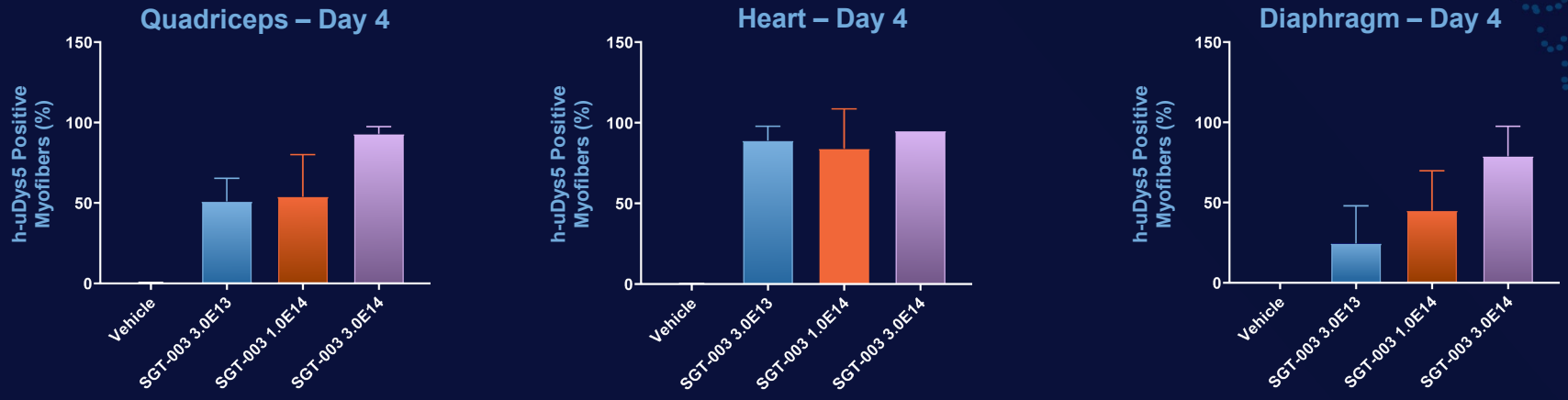


SOLID BIOSCIENCES

INSPIRE DUCHENNE SGT-003 Phase 1/2 Trial Dosing Initiated: Distinct U.S. and Global Regulatory Pathways Anticipated



Rapid AAV-SLB101 Transduction and Expression in mdx Mouse Model by Day 4



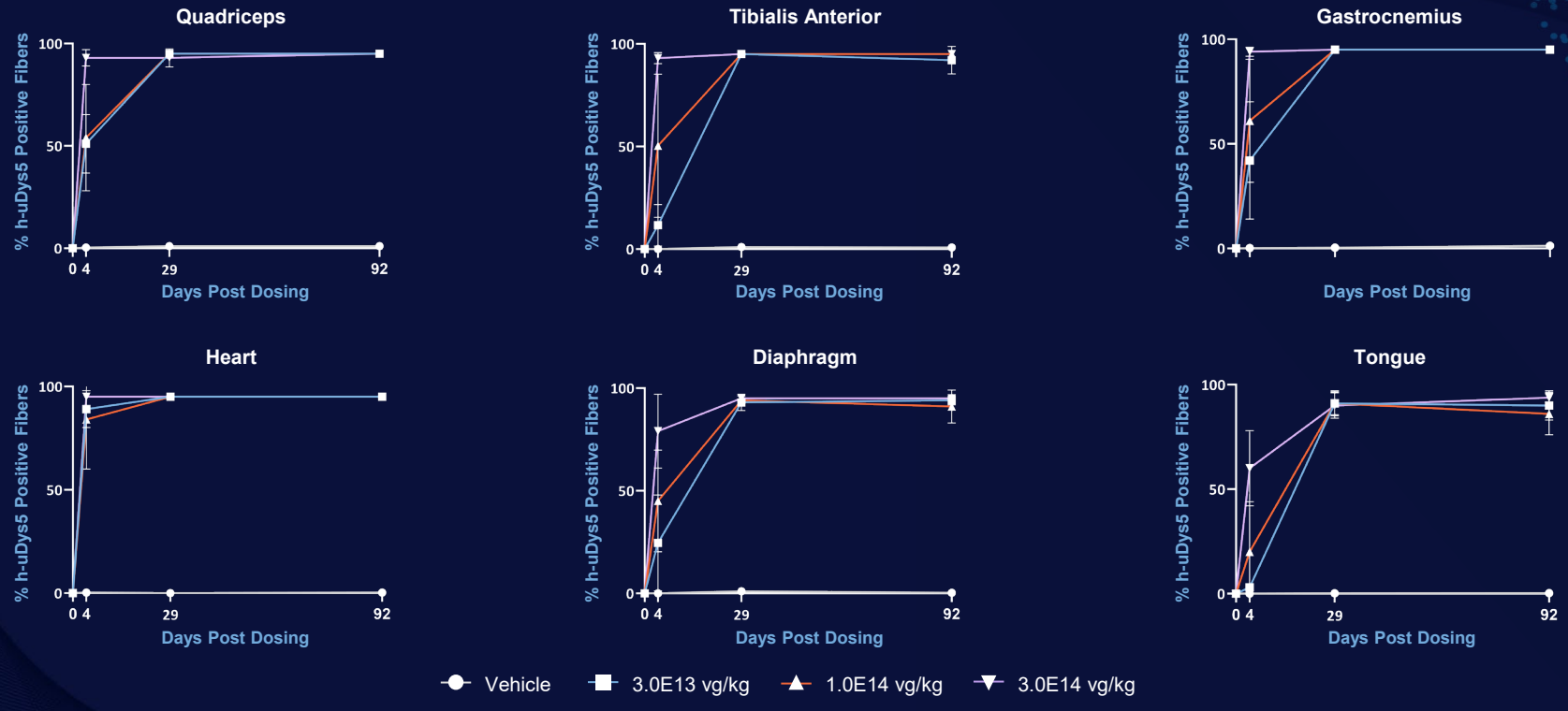
Observations

Robust microdystrophin expression levels, as assessed by h-uDys5+ myofibers in quadriceps, heart, and diaphragm, were evident by Day 4 post-AAV-SLB101 administration

h-uDys5, Solid's microdystrophin construct
N=5 per group

SOLID BIOSCIENCES

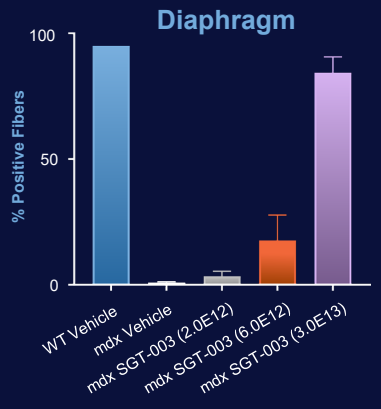
SGT-003 Showed Sustained Microdystrophin Expression in mdx Mouse Model

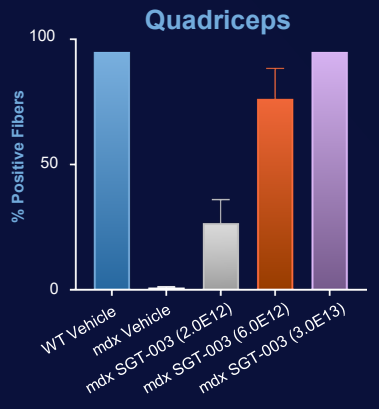


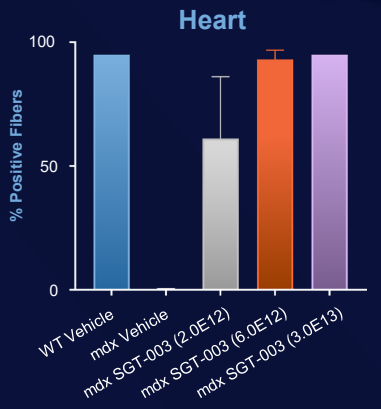
h-uDys5, Solid's microdystrophin construct
N=5 per group

High Microdystrophin Expression and nNOS Activity in Multiple Tissues at Low Doses in mdx Mouse Model

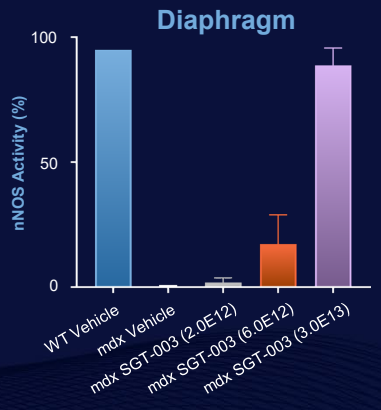
Microdystrophin

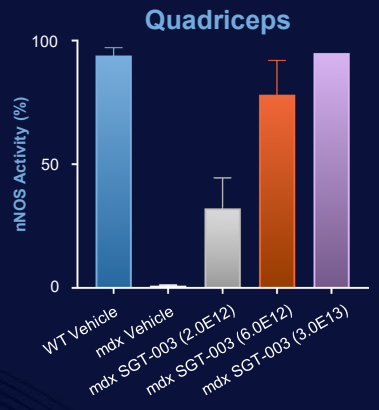






nNOS Activity





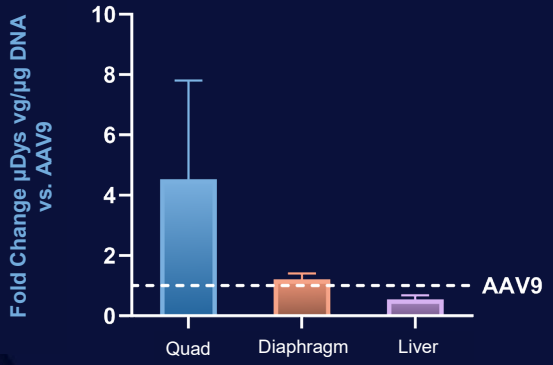
N=10 per group

SOLID BIOSCIENCES

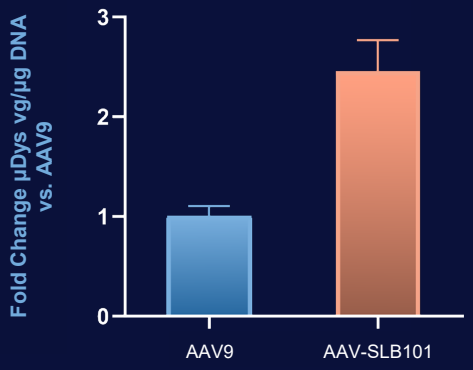
SGT-003 With AAV-SLB101 Capsid Demonstrated Superior Muscle Tropism vs AAV9

Positive Biodistribution and Expression Data Have the Potential to Translate Into Better Efficacy

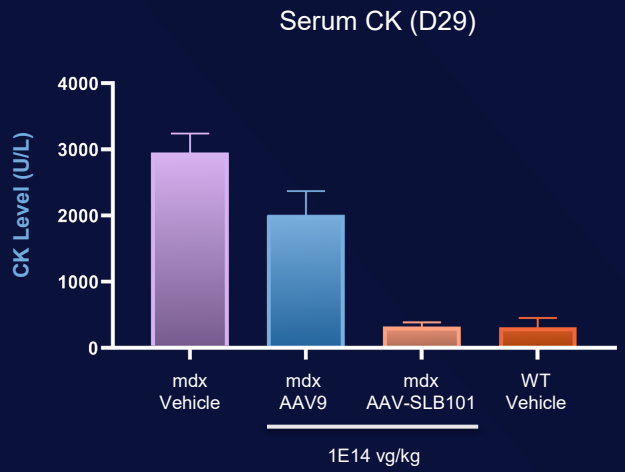
Tissue Specific Biodistribution in mdx Mouse



Robust µDys Expression in mdx Mouse



Reduced CK Levels in mdx Mouse

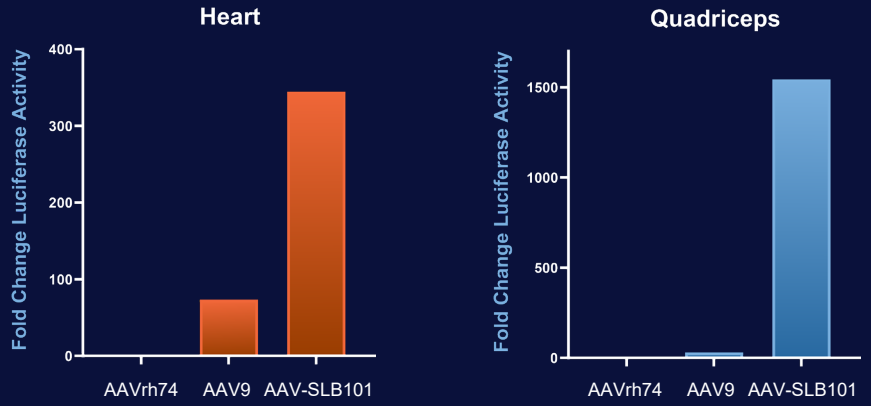


N=5 per group
Dose 1E14 vg/kg

AAV-SLB101 Exhibited Superior Protein Expression Profiles vs AAV-rh74 Across Muscle Tissues of NHPs and Mice

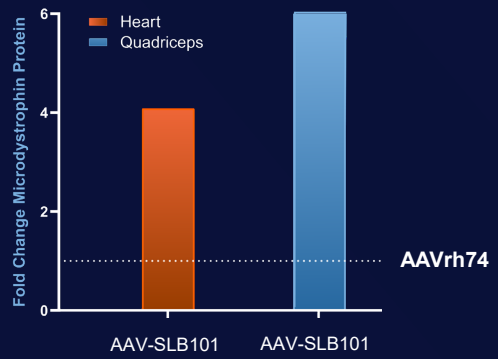
AAV-SLB101 vs AAV9 & AAV-rh74

CK8-Luciferase activity in mouse tissues at a low dose (2.0E12 vg/kg)[a]



AAV-SLB101 vs AAV-rh74

CK8-Microdystrophin protein expression by LC/MS in NHP tissues at clinical dose (1.0E14 vg/kg)[b]



Superior transgene expression profile of AAV-SLB101 in mouse and NHP muscle tissues justified candidate selection and IND-enabling studies with the AAV-SLB101-based therapeutic candidate SGT-003

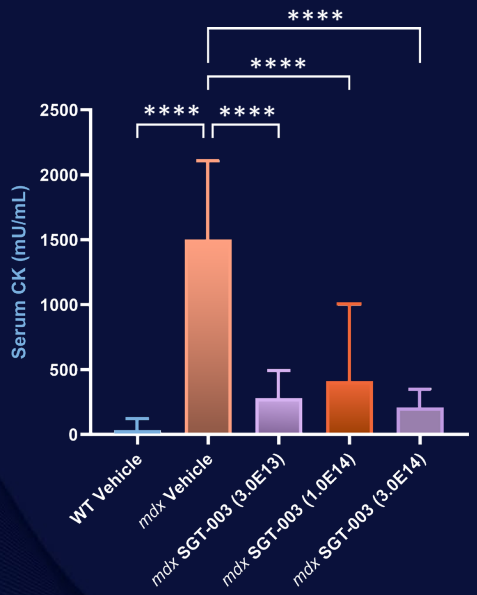
IND=investigational new drug; LCMS=liquid chromatography mass spectrometry

[a]N=5 for each tissue/capsid. [b]N=3 for AAV-SLB101. N=2 for AAV-rh74

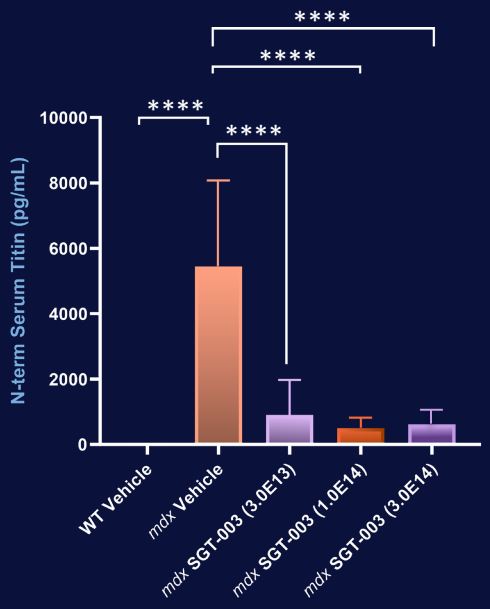
Data on file. Solid Biosciences. 2024.

Improved Serum Biomarkers of Muscle Membrane Integrity Seen in SGT-003-Treated *mdx* Mice at Doses ≥3.0E13 vg/kg

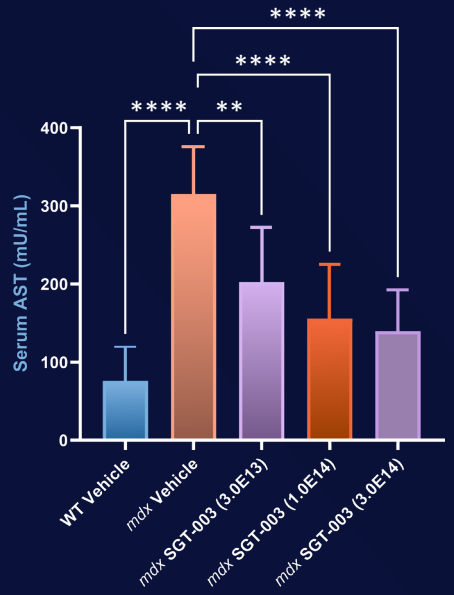
SERUM CK



SERUM TITIN

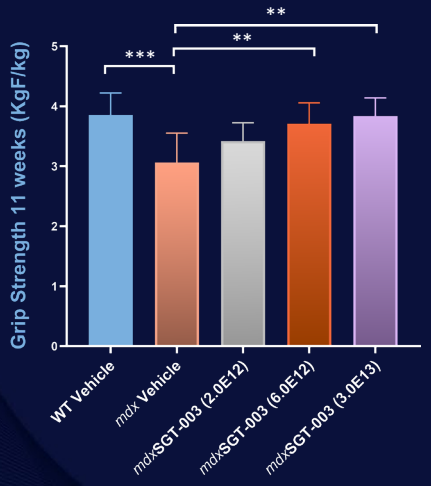


SERUM AST



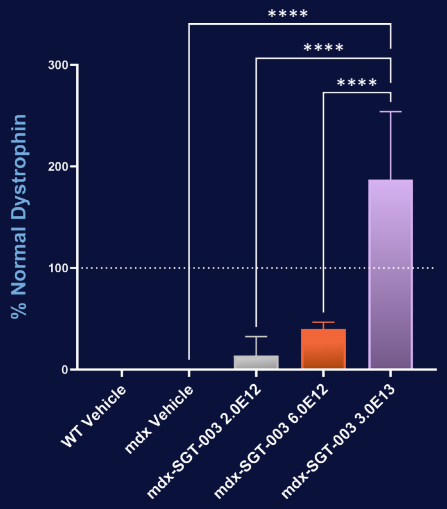
Significant Microdystrophin Expression and Functional Efficacy Observed in mdx Mouse Model at Low Doses (>6E12)

Grip Strength (11 weeks)

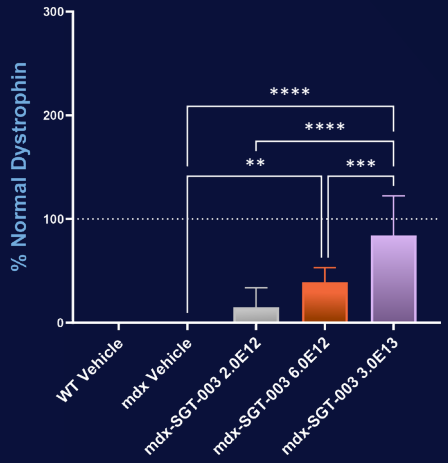


Mass Spectrometry - % Normal Dystrophin

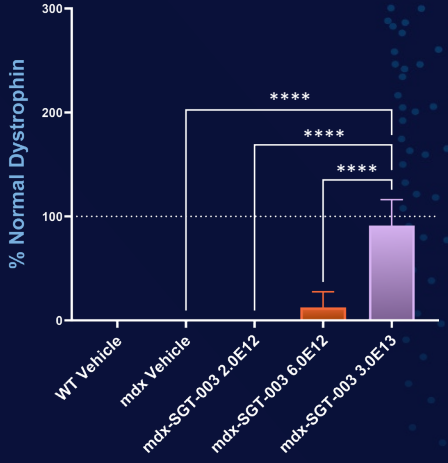
Heart



Quadriceps



Diaphragm



**p<0.005, ***p<0.0005, ****p<0.00005

Dotted line = 100% dystrophin threshold in adult skeletal muscle

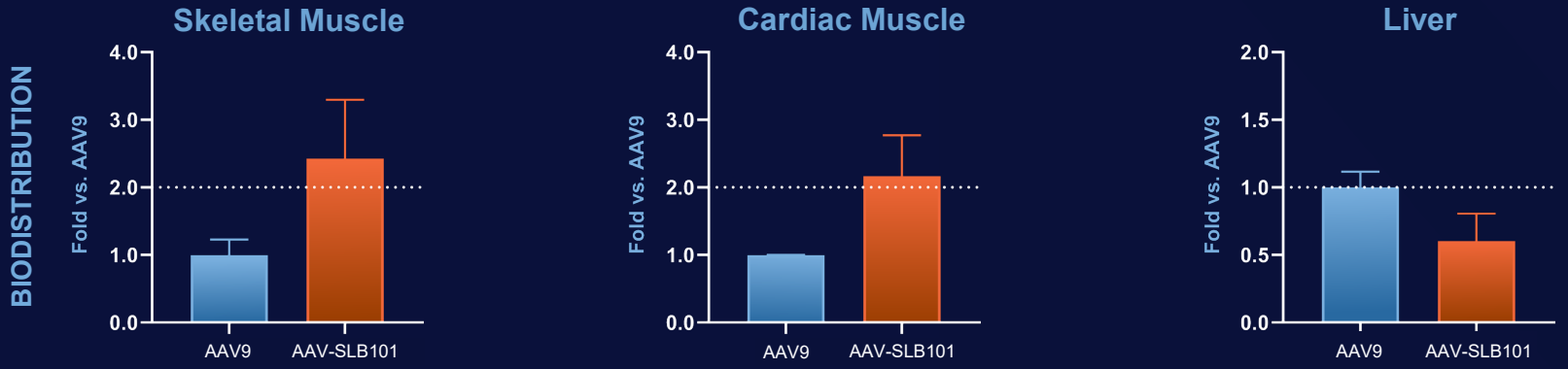
N=10 per group

SOLID BIOSCIENCES

NHPs Administered AAV-SLB101 Showed Improved Biodistribution in Cardiac and Skeletal Muscle With Decreased Hepatic Transduction as Compared to AAV9

- **Increased biodistribution to skeletal & cardiac muscle** correlated with increased transgene expression*
- **Reduced biodistribution in liver** suggests tissue de-targeting and potentially improved safety profile*

NHP IV Administration of AAV-SLB101 With Constitutive Promoter and Reporter Gene



*Average fold differences calculated from the five skeletal muscle tissues sampled, three regions of cardiac tissue sampled, and the single liver sample.

Dose 5e12 vg/kg

N=2 per group

SOLID BIOSCIENCES

GLP Toxicology NHP Study Showed SGT-003 Was Well Tolerated

NHP GLP TOX Study

3-month study

2 treatment groups
(1E14 vg/kg & 3E14 vg/kg)

n = 3/group

> 60 tissues evaluated including skeletal muscle, liver, brain

FINDINGS

- Well tolerated in both groups throughout the study
- No early mortality events, no unscheduled take downs
- No pathology findings: organ weight changes, macroscopic, or microscopic
- Liver enzyme levels comparable to vehicle at target clinical dose
- NHPs dosed at 3x planned first-in-human dose level (1E14 vg/kg)

SOLID BIOSCIENCES

INSPIRE DUCHENNE Clinical Trial Design: Ongoing SGT-003 Phase 1/2 Study

First-in-Human Open-Label, Single-Dose Study to Enroll a Minimum of 6 Patients

Objective | Design | Endpoints

Primary Objective

- To investigate the **safety and tolerability** of a single intravenous 1E14vg/kg dose of SGT-003

Secondary Objective

- To investigate the **efficacy** of a single intravenous dose of SGT-003

INSPIRE DUCHENNE

SOLID BIOSCIENCES

INSPIRE DUCHENNE Clinical Trial Design: Ongoing SGT-003 Phase 1/2 Study

First-in-Human Open-Label, Single-Dose Study to Enroll a Minimum of 6 Patients

Objective | **Design** | Endpoints

Design

Study includes **2 cohorts** based on age and weight at the time of signing the informed consent:

- Cohort 1: Participants 4 to < 6 years of age, ≤25 kg
- Cohort 2: Participants 6 to < 8 years of age, <30 kg

All participants are required to be on a stable dose of at least **0.5 mg/kg/day** of oral daily prednisone or **0.75 mg/kg/day** deflazacort for ≥12 weeks prior to entering the study

All participants must be ambulant and have a diagnosis of DMD with a documented dystrophin gene mutation confirmed by genetic testing at screening.

INSPIRE DUCHENNE

SOLID BIOSCIENCES

INSPIRE DUCHENNE Clinical Trial Design: Ongoing SGT-003 Phase 1/2 Study

First-in-Human Open-Label, Single-Dose Study to Enroll a Minimum of 6 Patients

Objective | Design | **Endpoints**

Primary Endpoint

- Incidence of treatment-emergent adverse events (AEs) through Day 360

Secondary Endpoints

- Change from baseline of microdystrophin protein levels at Day 90 and 360
- Change from baseline in the NSAA score at Day 360
- Change from baseline in stride velocity 95th percentile (SV95C) at Day 360

INSPIRE DUCHENNE

SOLID BIOSCIENCES

Cardiac Lead Program

Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT)

SOLID BIOSCIENCES

CPVT

Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT): a Fatal Disorder in a Young Population

Affected Population	PREVALENCE	ESTIMATED
	1:10,000 people[1]	~33,000 patients in the US

Cause

CASQ2 & RYR2 proteins regulate cardiac calcium (Ca^{2+}), important for electrical conduction and cardiac contraction / relaxation

Postulated Mechanism: Mutations in RYR2 or CASQ2 genes disrupt Ca^{2+} release into the cytoplasm triggering abnormal contraction and relaxation leading to arrhythmias

Solid Approach

AAV-delivered, CASQ2 transgene with cardiac-selective promoter designed for safe expression utilizing optimized transient transfection manufacturing process

Clinical Presentation and Unmet Need

SIGNS & SYMPTOMS

- Most commonly presents as syncope events or cardiac arrest
- Quality of life severely impacted. Risk of spontaneous arrhythmias and or sudden death
- Poor Prognosis: ~40% mortality within 10 years of diagnosis[2]

AGE OF ONSET

- Typically identified in younger patients (mean onset between 7-12 y/o)

STANDARD OF CARE

- No available targeted therapies to address underlying disease cause

RYR2=Ryanodine receptor, CASQ2= Calsequestrin 2

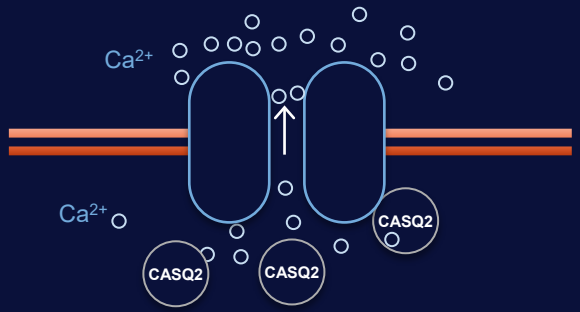
1. Napolitano, et al. 2022 2. Sumitomo, 2016

Rationale for CASQ2 Overexpression in RYR2 CPVT

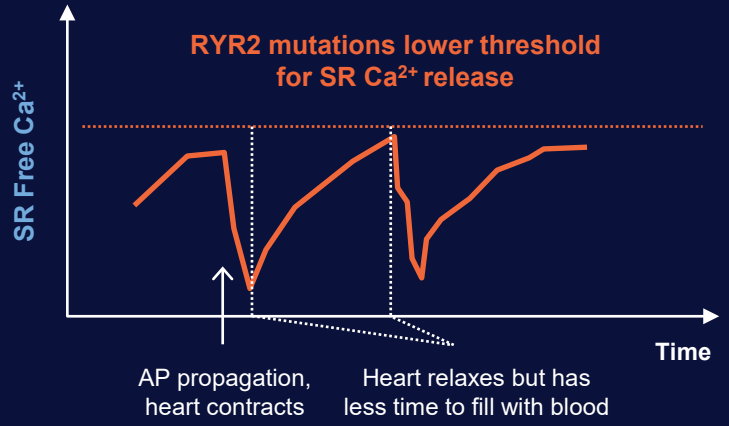
CASQ2 Overexpression Leads to Increased Ca^{2+} Buffering to Counteract Ca^{2+} Sensitivity of RYR2 Mutant

RYR2 Mutation-Related CPVT

Mutations in RYR2 make the channel more sensitive to SR Ca^{2+} levels, resulting in early release of Ca^{2+} into the cytoplasm and the heart contracting when it should be filling with blood in diastole



Arrhythmia



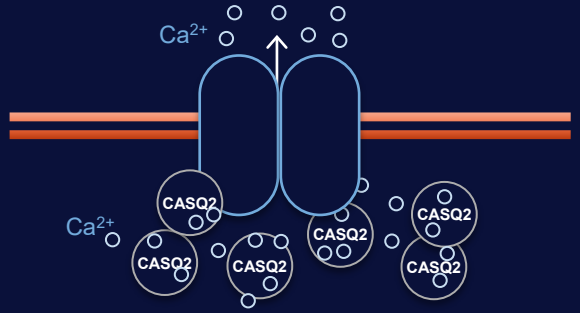
RyR, Ryanodine Receptor; SR, sarcoplasmic reticulum; AP, action potential; Ca^{2+}, calcium
Sources: Proprietary data from Priori Lab

Rationale for CASQ2 Overexpression in RYR2 CPVT (cont.)

CASQ2 Overexpression Leads to Increased Ca^{2+} Buffering to Counteract Ca^{2+} Sensitivity of RYR2 Mutant

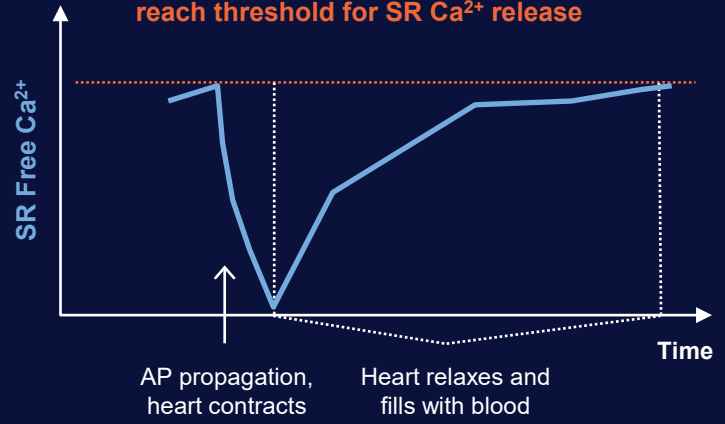
RYR2 Mutation-Related CPVT + Overexpressed CASQ2

Increased CASQ2 enhances Ca^{2+} buffering and helps stabilize RYR2 in the closed state, which extends diastole until the typical time of contraction, allowing the heart to fill fully



Normal Rhythm

Overexpressed CASQ2 increases time to reach threshold for SR Ca^{2+} release



RyR, Ryanodine Receptor; SR, sarcoplasmic reticulum; AP, action potential; Ca^{2+}, calcium
Sources: Proprietary data from Priori Lab

RYR2 CPVT Transgenic Mouse Model Used To Support Proof of Concept For AAV Gene Delivery of Human CASQ2

RYR2 Transgenic Mice Have An Arrhythmogenic Phenotype Upon Challenge With β-Adrenergic Agents[1]

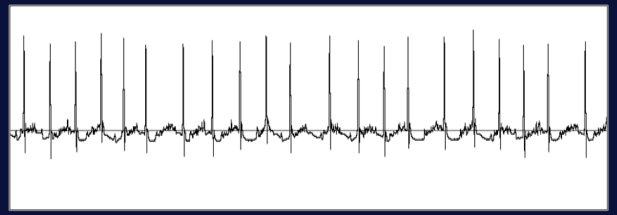
WT Mice

IP dose epinephrine & caffeine

RYR2 Transgenic Mice

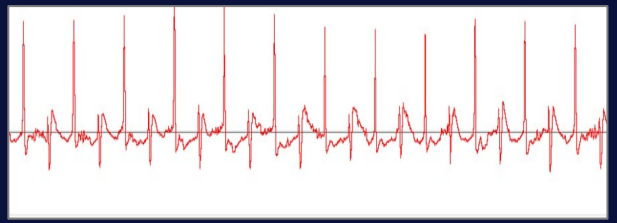
IP dose epinephrine & caffeine

Wild Type



Normal heart rhythm **in WT background strain animals**

RYR2 Transgenic



Polymorphic and/or bidirectional arrhythmic morphology **in transgenic animals**

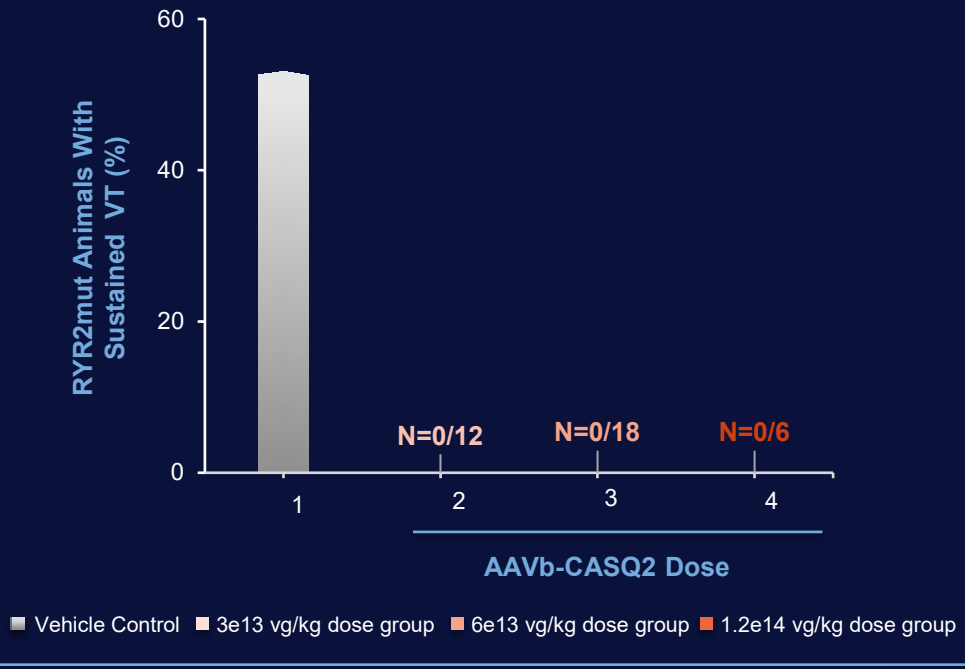
1. Cerrone, et al. 2005

Elimination of Arrhythmias in Multiple Disease State Models

Data Suggests CASQ2 Augmentation Was Well Tolerated & Highly Protective in RYR2- & CASQ2-Related CPVT Arrhythmias

RYR2-related CPVT Mouse

At 12 weeks old, **none** of the **RYR2 mutant mice** treated with AAVb-CASQ2 gene therapy exhibited arrhythmias[1]



1. Priori Lab, unpublished data

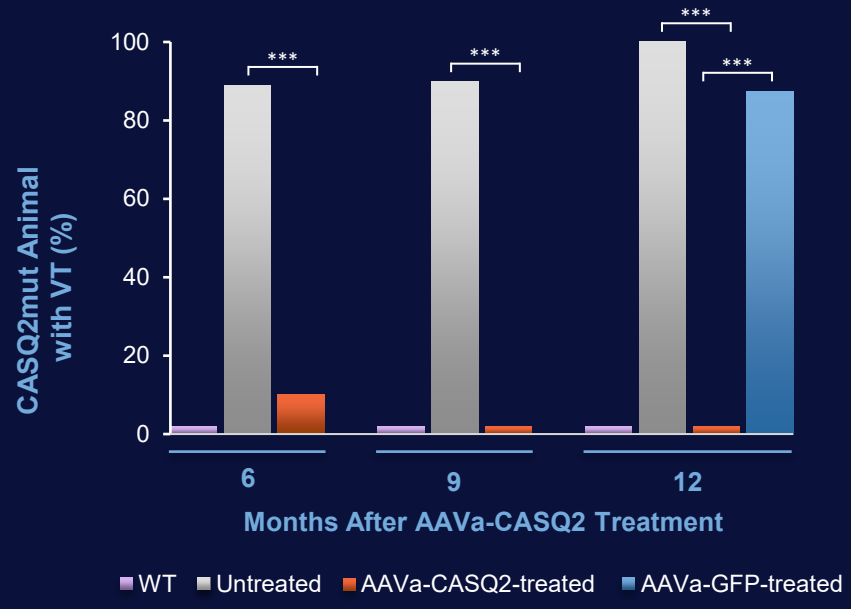
Elimination of Arrhythmias in Multiple Disease State Models

Data Suggests CASQ2 Augmentation Was Well Tolerated & Highly Protective in CASQ2 & RYR2 CPVT Arrhythmias

CASQ2-related CPVT Mouse

Significantly fewer **CASQ2 mutant mice** experienced arrhythmias 6-12 months after AAVa-CASQ2 gene therapy[1]

40-50% transduction, achieved in both neonates and adult mice, prevented propagation of triggered beats[1]



***P<0.001, AAVa-CASQ2–treated vs untreated and AAVa-CASQ2–treated vs AAVa-GFP–treated

1. Denegri, et al. 2014

Pipeline Programs

BAG3

SOLID BIOSCIENCES

BAG3

Attractive Indication, Clear Mechanistic Rationale, High Unmet Need & Significant Market Size

Affected Population	PREVALENCE	ESTIMATED	ESTIMATED
	2-4% DCM Cases[1]	~29,000 patients in the US	~33,000 patients in the EU

Cause

BAG3 mutations lead to reduced BAG3 protein leading to dilated cardiomyopathy (DCM)

Postulated mechanism: Decreased BAG3 protein leads to heat shock protein dysfunction and a build-up of dysfunctional proteins in the sarcomere, causing myofilament damage and heart failure

Solid Approach

Delivering a codon optimized BAG3 gene with a cardiac-selective promoter utilizing transient transfection manufacturing process

Clinical Presentation and Unmet Need

SIGNS & SYMPTOMS

- Most common presentation is dyspnea (but can be sudden death)
- Activities of daily life are severely impacted
- Adverse long-term prognosis, approximately 25% at one year and ~50% at five years experience severe cardiac event, intervention, or death[1]

AGE OF ONSET

- DCM caused by mutations in BAG3 is characterized by high penetrance in carriers >40 years of age and a high risk of progressive heart failure[1,2]

STANDARD OF CARE

- No approved therapies address underlying cause of disease

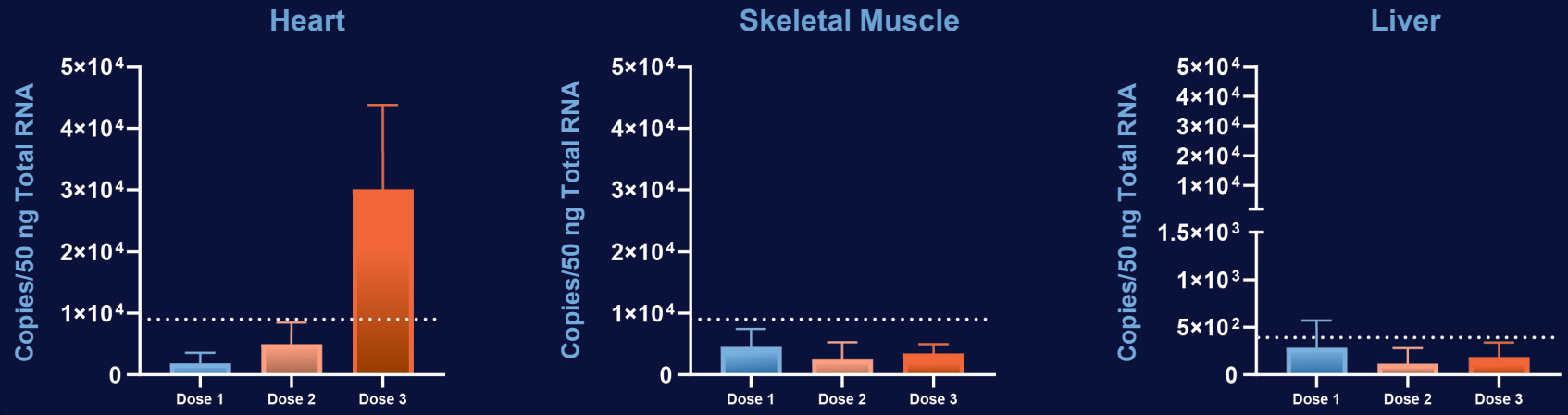
1. Dominguez, et al. 2018, 2. Shaw, et al. 2018

SOLID BIOSCIENCES

Cardiac-Selective Expression of Human BAG3 in Cardiac-Specific BAG3 Mouse Model

Expression of Human Transgene mRNA Is Below Endogenous BAG3 mRNA Levels in Off-Target Tissues

BAG3 Cardiac-Specific Knockout (cKO) Mouse IV Administration of AAV with Cardiac Promoter and Human BAG3 Transgene



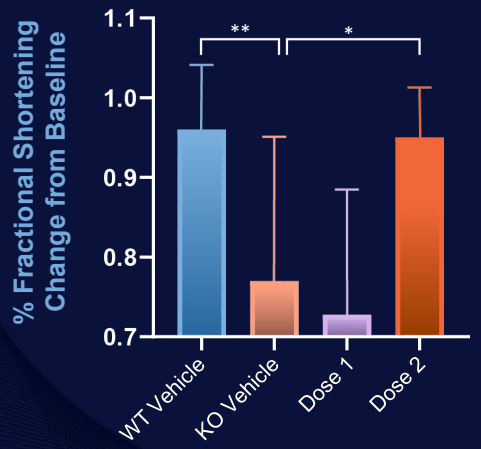
Dotted line = endogenous mouse BAG3 mRNA levels in each tissue; cKO = conditional knockout

Exploratory Efficacy Study Suggests Improved Cardiac Function in BAG3 cKO Mouse Model

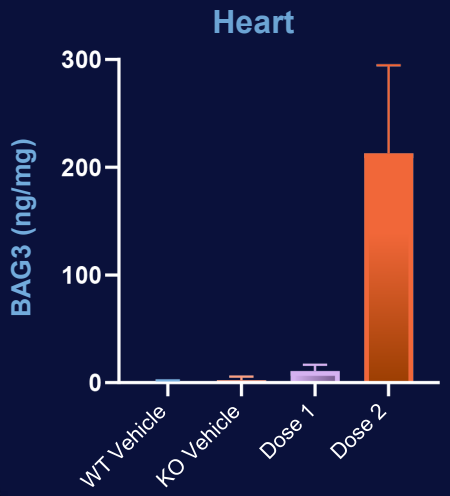
Mouse Data Support Continued Development of AAV-Mediated Gene Delivery of Human BAG3

cKO of BAG3

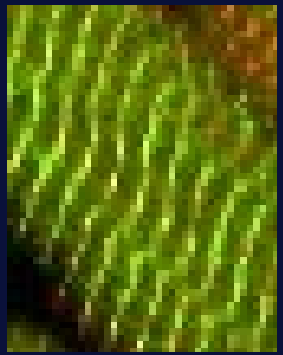
Dose-dependent Improvement in Cardiac Function



Dose-dependent Expression of Human BAG3 Protein



Human BAG3 Protein Localized to Z-line in Cardiomyocytes



Green= BAG3; Red= a-actinin

*p <0.05 One way ANOVA, Tukey's multiple comparisons test

N=5-14

SOLID BIOSCIENCES

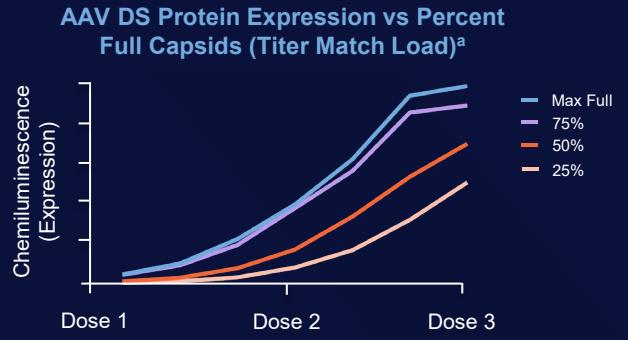
Platform Technologies

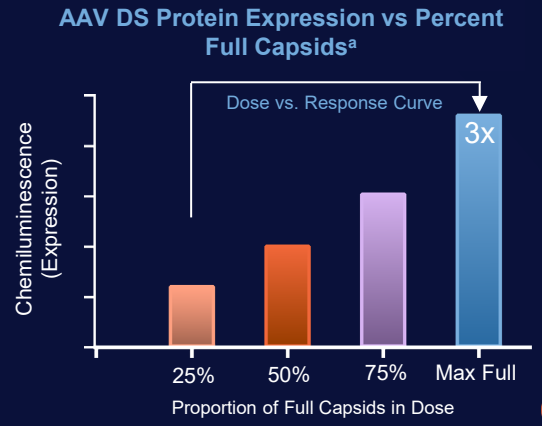
SOLID BIOSCIENCES

Full/Empty Capsid Ratio Can Impact Transduction and Expression of AAV Products

~3-Fold Difference in Chemiluminescence (Expression) Based on Full/Empty Percentage

- Transducing C2C12 cells with an AAV luciferase construct allowed chemiluminescence to act as a readout of expression
- Keeping ddPCR titer constant and serially diluting with empty capsids demonstrated that expression was impacted at constant dose
- **Maximizing the percentage of full capsids has the potential to improve expression and safety of an AAV product**





ddPCR=Droplet Digital PCR.

[a]AAV-luciferase diluted with empty AAV capsids to yield theoretical 25%-100% full capsids with a series of dilutions based off initial gene of interest titer. N=3 per sample.

Data on file. Solid Biosciences. 2024.

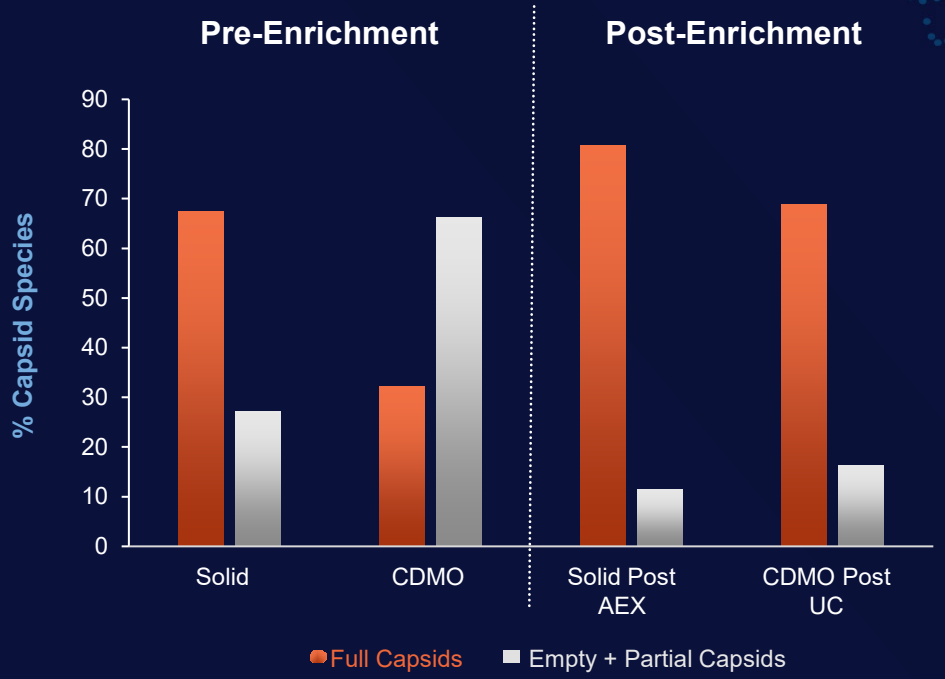
SOLID BIOSCIENCES

Solid's Manufacturing Platform Has Potential to Challenge Industry Yields and Redefine Full/Empty Capsid Purity

Significant Increase in Yields and Continued Improvements in Full/Empty Ratios Seen at Research Scales*

Yield & Quality Performance

Solid's pre-enrichment capsids (full vs. empty + partial) superior to leading CDMOs



*Data on file, currently at 2L scale in process development (PD), DMD clinical program currently at 1000L

Anticipated Near Term Milestones

	Program	Milestone (anticipated)	Timing
Neuromuscular	SGT-003 for Duchenne	Patient dosing commenced	✓
		Submit multiple CTAs for global trial (already authorized in Canada)	Ongoing
		Initial phase 1/2 data (safety, microdystrophin expression & functional data)[1]	Q4 2024
Cardiac	SGT-501 for CPVT	Preclinical studies in NHP and mouse	Ongoing
		Planned submission of RYR2 IND	1H 2025
Capsids	AAV-SLB101	First-in-human data	Q4 2024
	Capsid Library (multiple capsids)	Complete rounds of NHP, mouse, and pig studies	Ongoing
Pipeline	Multiple Pipeline Assets	BAG3 preclinical studies, TNNT2 NHP & mouse studies, RBM20 preclinical work	Ongoing

1. Initial safety and 90-day expression and functional data